UNIVERSITY OF LONDON
GALTON LABORATORY FOR NATIONAL EUGENICS

EUGENICS LABORATORY LECTURE SERIES. VIII

Tuberculosis, Heredity and Environment

BY

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CAMBRIDGE UNIVERSITY PRESS
C. F. CLAY, MANAGER
LONDON: FETTER LANE, E.C. EDINBURGH: 100, PRINCES STREET
H. K. LEWIS, 136, GOWER STREET, LONDON, W.C.
WILLIAM WESLEY AND SON, 28, ESSEX STREET, LONDON, W.C.
CHICAGO: UNIVERSITY OF CHICAGO PRESS
BOMBAY, CALCUTTA AND MADRAS: MACMILLAN AND CO., LIMITED
TORONTO: J. M. DENT AND SONS, LIMITED
TOKYO: THE MARUZEN-KABUSHIKI-KAISHA

1912

Price One Shilling net

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BEING A LECTURE DELIVERED AT THE GALTON LABORATORY FOR NATIONAL EUGENICS, MARCH 12, 1912

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TUBERCULOSIS, HEREDITY AND ENVIRONMENT

If we study the history of medical opinion with regard to phthisis, which may readily be done in the recent very able paper by Dr. Bullock and Mr. Greenwood,[1] we find that in the past much weight has been given to the constitutional factor. To the great medical writers of the earlier part of last century the soil meant everything and the germ little at all, possibly—as some of their critics would now say—because they knew nothing about it. With the discovery of the tubercle bacillus the pendulum swung at once to the other side—the idea of infection dominated everybody, and the views of the clinician were replaced by those of the laboratory investigator, who could determine, far more dogmatically than the clinician, the presence of tubercle. The result was an immediate neglect both of the hereditary factor and of the environmental factor. The origin of phthisis was infection, and the battle against phthisis was summed up in the destruction of the tubercle bacillus or of such environments as were supposed to encourage its existence. The importance of the discovery of Koch cannot be overrated, but, as a result of it, a very definite line of action was taken without any adequate scientific inquiry into the relative importance :

(i) of the hereditary factor ;
(ii) of the environmental factor ;
(iii) of the liability to infection.

[1] *Royal Soc. Medicine*, April, 1911.

Almost every fact recorded can be interpreted without cautious inquiry as illustrative of the importance of any one of these three factors, according to the *a priori* conviction of the interpreter. For example, let us look at two or three examples of 'family phthisis'.

Figs. I and II give two pedigrees due to Klebs. At first sight Klebs' pedigrees seem to mark inheritance, but it is equally open to the environmentalists to assert that in such cases the family has occupied the same house or lived in the same village. The infectionist can assert equally well that the infection has been handed down from parent to child, generation by generation. If you examine Klebs' cases a little more closely you will find 16 cases of marriage of phthisical individuals, and only two cases of *both* husband and wife being phthisical. In fact, 62% of the tuberculous members lived to adult life, of whom 45% married; only 12·5% of their mates, but 79% of their offspring were tuberculous. If the phthisis in the children be due in the first place to infection from parents or to a common environment, we should anticipate that more than $\frac{1}{8}$ of the mates of phthisical individuals would themselves be phthisical. This in Germany is very nearly the number of phthisical persons occurring in the population at large. In the third pedigree (Fig. III, p. 6) there are no phthisical mates, and the tuberculosis has skipped now one and now two generations. It is less easy to believe that the infection factor here is the principal source of the heavy mortality, as the members of the stock have lived in widely separated homes and under very diverse conditions, flying from the family curse. The environmental factor can hardly be of significance. I do not propose to base any argument on these pedigrees—that would

FIG. 42 Klebs' Case (i.)

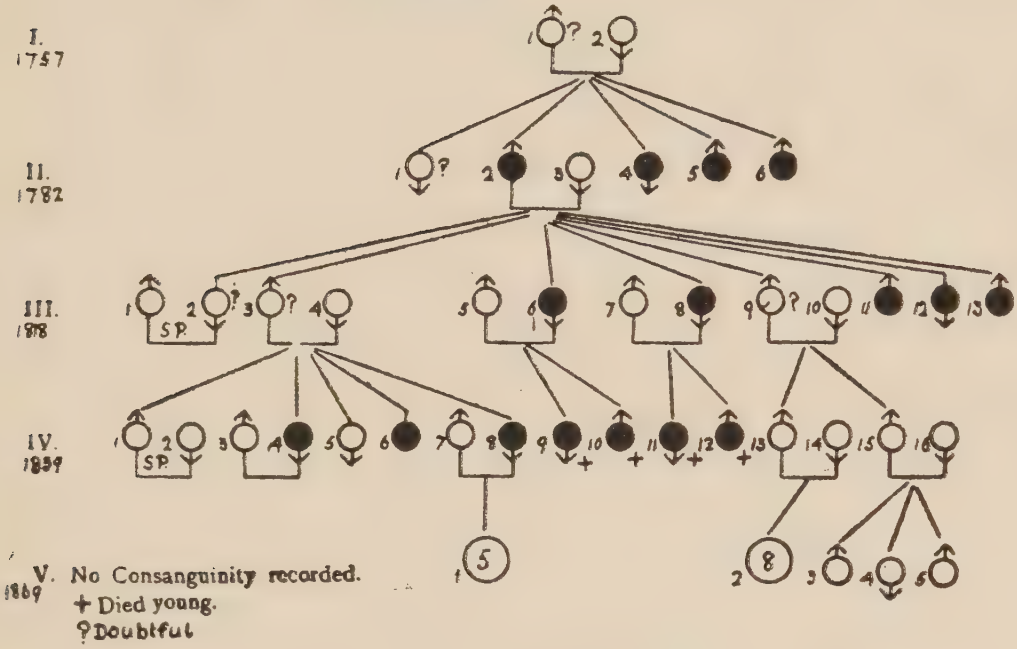


Fig. I. From the *Treasury of Human Inheritance.*

FIG. 43 Klebs' Case (ii.)

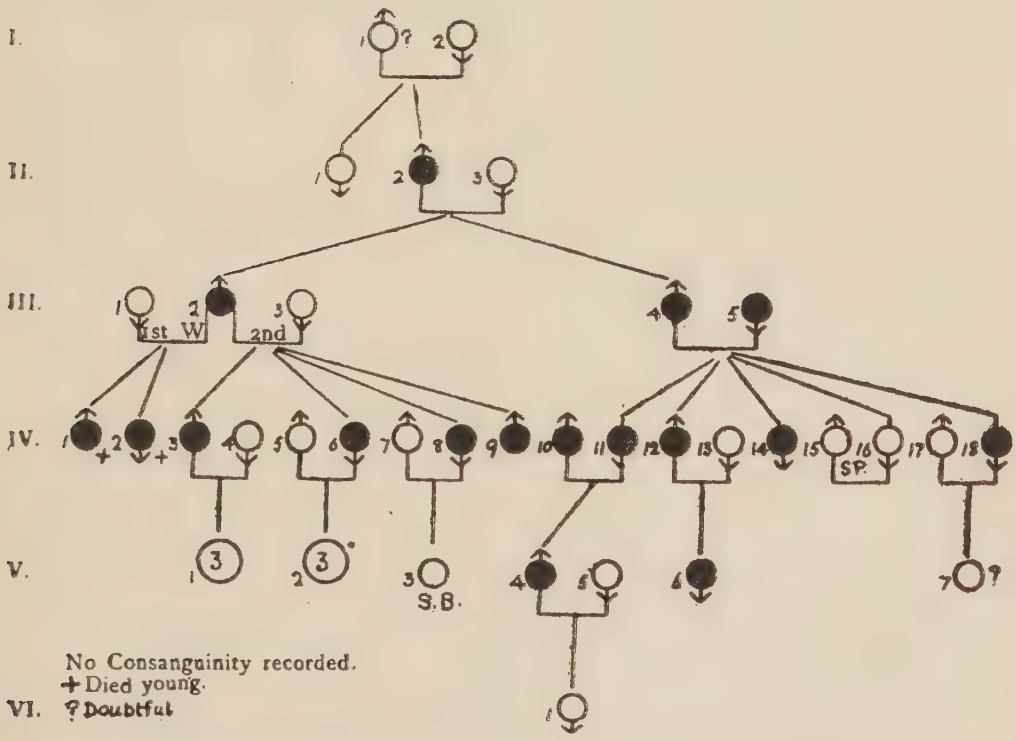


Fig. II. From the *Treasury of Human Inheritance.*

only be legitimate if I were basing percentages on many hundreds—but I use them only to emphasize the nature of the problems which arise, i. e. :

(*a*) We have to inquire whether persons living habitually in the same environment and with practically the

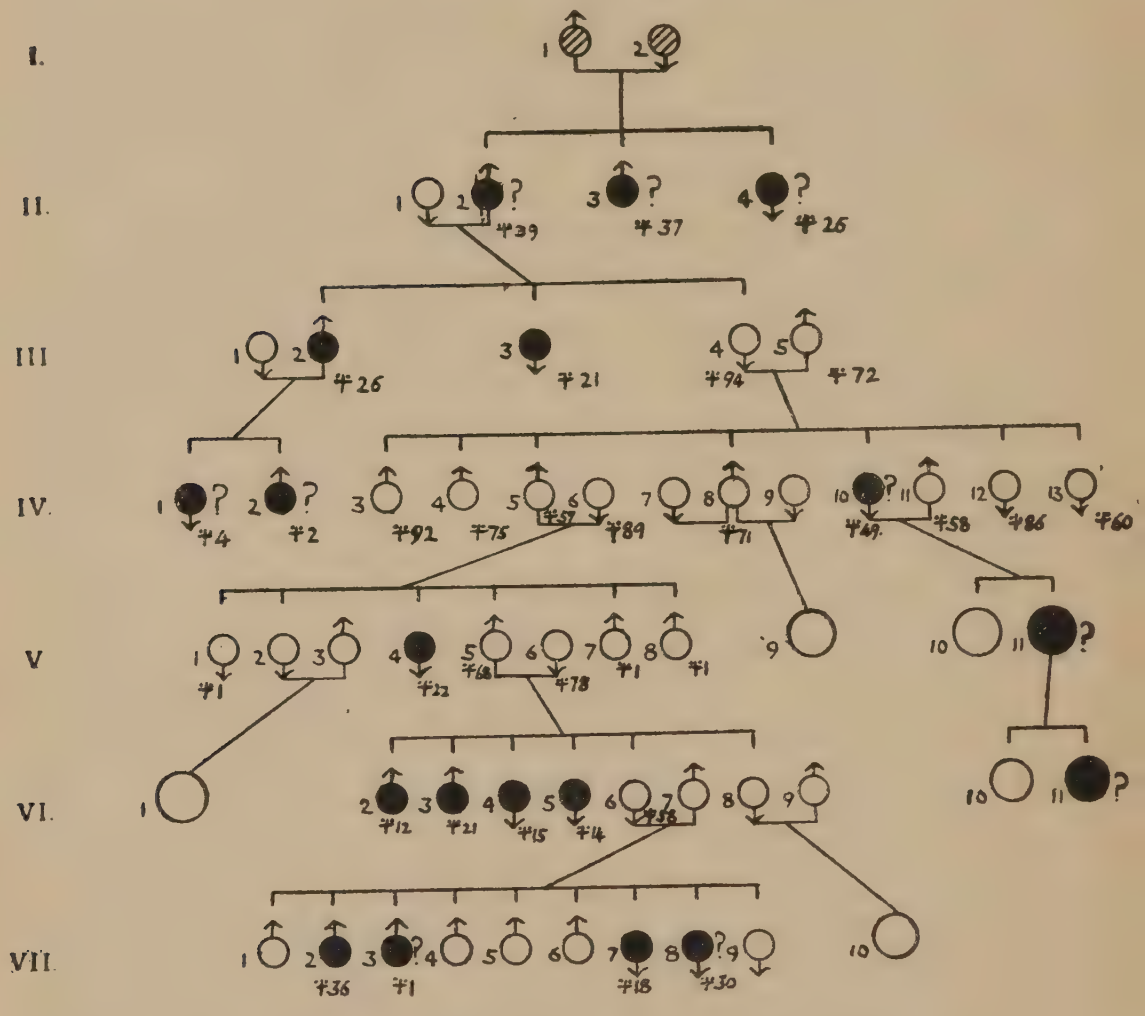


Fig. III. Family Phthisis : Pearson's Case.

same risk of infection have the same chance of developing phthisis whatever be their stock. There is no closer relation than that of husband and wife. Is the mate of a phthisical person as likely to acquire the disease as the offspring of such a person ?

(*b*) If we find numerous cases of phthisis in a stock,

does the disease reappear in the members who have had no contact with the phthisical members? or again, do we find it skipping generations?

These problems are not easy problems to answer;[1] they are surrounded with very subtle statistical difficulties, but I have no patience with those writers who, without entering into any analysis of such pedigrees as I have shown you, dismiss them with the remark that they only represent 'family infection'. Do we meet with risk of infection only in the family? If not, what is the relative importance of the risks we run within the family and outside it? All these are problems which want a thorough thrashing out, but which have been largely side-tracked by the idea that infection is the only factor worthy of our consideration. On these topics I can only touch briefly to-night, but I should like to bring some remarkable points to your notice.

In association with the Biometric Laboratory much statistical work has recently been done on phthisis. In the first place we have tried to appreciate the extent of marital infection in tuberculosis.

From the family records in the Laboratory, if we leave out the 0·5% of cases in which the condition of either husband or wife was unknown, and add to the tuberculous the 0·6% of cases returned as doubtful, we have on the basis of 1,000 marriages in the professional classes the results shown in Table I. This indicates that in 1,000 marriages there are 55 wives and 49 husbands tuberculous, and of these 6 cases of tuberculous hus-

[1] The work of Dr. Chalmers in Glasgow is undoubtedly on the right lines, but as represented in the Reports of the Local Government Board for Scotland on the *Administrative Control of Pulmonary Phthisis in Glasgow*, 1911, the necessary information just at the critical points appears to be omitted.

band and wife. How many should we expect on the basis of pure chance? Only three pairs [i.e. $1000 \times \frac{55}{1000} \times \frac{49}{1000} = 2{\cdot}695$]. There is clearly, therefore, a transfer of six individuals in all. On the scale of

TUBERCULOSIS IN HUSBAND AND WIFE.

TABLE I.

PEARSON: Professional Classes.

Actual:

		Husband Not T.	T.	Totals.
Wife	Not T.	902	43	945
	T.	49	6	55
Totals		951	49	1,000

Correlation 0·24

Chance:

		Not T.	T.	Totals.
Wife	Not T.	899	46	945
	T.	52	3	55
Totals		951	49	1,000

Transfer

+3	−3
−3	+3

relationship the correlation is 0·24. Shall we argue that to this extent the phthisical individuals infect their mates? It is better to investigate further before we do more than note the fact that in the professional classes there is a somewhat greater chance of the tuberculous having a tuberculous mate. Dr. Goring has repeated a similar investigation on the poorer classes, such as provide the bulk of our criminals. He has dealt first with the well-to-do and prosperous of the poorer classes, as shown in Table II. The transfer here is curiously enough exactly the same, but since the percentage of the tuberculous has risen the result is not so close.

Actually the pairs of tuberculous mates observed are rather less than double the chance number of such pairs. The correlation has fallen to 0·16. In Table III

TABLE II.

GORING: Well-to-do and Prosperous Poor.

Actual: *Husband.*

		Not T.	T.	Totals.
Wife	Not T.	885	50	935
	T.	58	7	65
	Totals	943	57	1,000

Correlation 0·16

Chance:

		Not T.	T.	Totals.
Wife	Not T.	882	53	935
	T.	61	4	65
	Totals	943	57	1,000

Transfer

+3	−3
−3	+3

TABLE III.

GORING: Poor of all classes.

Actual:
= Chance.

Husband

		Not T.	T.	Totals.
Wife	Not T.	873	64	937
	T.	59	4	63
	Totals	932	68	1,000

Correlation 0·00

Transfer

0	0
0	0

we have Goring's results for tuberculosis in husband and wife for the poor of all classes—the distribution actually observed is to a unit the same as the chance distribution—the correlation is zero, or there appears to be no greater chance of the mate of a tuberculous person being tuberculous than of any other person. There appears to be matter here to lead us to pause. As we go

downward in the social scale the chances that a tuberculous husband will have a tuberculous wife diminish. This is a remarkable state of affairs if it has to be explained by infection! Pope has collected statistics of phthisis in husband and wife from 40 medical publications. If all the 41,786 couples dealt with by Pope be clubbed together, as in our Table IV, we see the total

TABLE IV.

POPE: from Records of upwards of 40,000 Tuberculous Cases.

Actual:

		Husband Not T.	T.	Totals.
Wife	Not T.	737	134	871
	T.	98	31	129
Totals		835	165	1,000

Correlation 0·17
Corrected 0·31

Chance:

		Not T.	T.	Totals.
Wife	Not T.	727	144	871
	T.	108	21	129
Totals		835	165	1,000

Transfer

+10	−10
−10	+10

transfer of individuals has now risen from 6 to 20; but this increase does not mean a higher correlation, for the percentage of tuberculous in Pope's data is *double* that of our English data, and the actual frequency is only 1·5 times, not *twice*, the chance frequency in the case of both husband and wife being tuberculous. The correlation is 0·17,[1] but this is not directly comparable with our previous results, for they were based on *random* samples of the populations dealt with: here, every

[1] The mean of the values of the 40 Tables treated separately given by Pope is 0·16, and agrees closely with the 0·17 obtained by combining them.

husband and wife has been selected because they were parents of at least one tuberculous child. If the view be held that tuberculosis is not hereditary, then although we have selected the parents of tuberculous children we should not very much increase the value just found, for the infection factor would only be of the order 0·17 (between parent and child also). But if we accept the hereditary factor, then the correction needful will send up the relationship between husband and wife to something like 0·30. These results as a whole seem to indicate that the average value of the correlation in phthisis between husband and wife must lie between 0·20 and 0·30, but that it decreases as we pass from the more highly educated to the lower classes.

We have to seek an explanation of this matter, and the first light we can throw on it is to find the resemblance of husband and wife in other characters than tuberculosis. I take the extremes : first, insanity, where we cannot imagine any infective relation, and then alcoholism, where we might suppose mutual influence to be great. In Table V we see the remarkable fact that the number of pairs of insane husbands with insane wives is more than double their chance frequency. The correlation of insanity in husband and wife is 0·24, exactly what we found for tuberculosis in material also drawn from the professional classes. In this table for insanity I have included among the insane some individuals having night terrors, or markedly neurotic. If these be included with the normal, the correlation rises to 0·30, or appears as large as the average value of Pope's data for tuberculosis. I now turn to alcoholism as obtained by Dr. Goring for upwards of 1,400 cases. Instead of a transfer of 14 individuals in the 1,000 we now

find one of 102 (see Table VI), and the correlation has risen to the high value of 0·70! In other words, the infection of tuberculosis between husband and wife is of a wholly different order to the infection of alcohol! But if the 0·25 resemblance of husband and wife in the case of tuberculosis is to be attributed to infection, what shall we say of the 0·25 in the case of insanity?

TABLE V. INSANITY.

PEARSON: Professional Classes.

Actual:

		Husband Not I.	I.	Totals.
Wife	Not I.	856	71	927
	I.	60	13	73
Totals		916	84	1,000

Correlation 0·24

Chance:

		Not I.	I.	Totals.
Wife	Not I.	849	78	927
	I.	67	6	73
Totals		916	84	1,000

Transfer

+7	−7
−7	+7

Can we not account for both on a like basis? It has been found impossible at present to define the constitution peculiarly liable to tuberculosis. Galton attempted it with composite portraits of the tuberculous, but it is difficult to describe verbally the facial differentiation, if any, of this composite.

Yet there are several mental characters of the phthisical which will be familiar to some of my audience, notably in many cases a certain strenuousness and keenness. It is conceivable that phthisical stocks are to a certain extent sympathetic to each other, and that

there is an actual sexual selection of those likely to become tuberculous. In the same way eccentric and mentally ill-balanced stocks have an attraction for each other. Sexual selection, as measured by the resemblance of husband and wife, is now a well-recognized fact. In almost every character husband and wife have a degree of resemblance almost equal to that of first cousins. Nobody supposes that husband and wife

TABLE VI. ALCOHOLISM.

GORING: Poorer Classes.

Actual:

		Husband Not A.	A.	Totals.
Wife	Not A.	711	194	905
	A.	18	77	95
Totals		729	271	1,000

Correlation 0·70

Chance:

		Not A.	A.	Totals.
Wife	Not A.	660	245	905
	A.	69	26	95
Totals		729	271	1,000

Transfer

+51	−51
−51	+51

select each other by the length of their forearms or by their tone of voice; nevertheless, their forearms and tone of voice are more alike than would be the case if pairs of men and women were drawn at random from the community. In the accompanying Table VII, I have put together a number of the results for sexual selection so far obtained in the Biometric Laboratory.

Now several conclusions may be safely drawn from this table:

Firstly, we see that husband and wife resemble each

TABLE VII. ASSORTATIVE MATING IN MAN.

Physical Characters.

Character.	Correlation.	Authority.	Material.
Eye Colour	·26	Pearson	Galton.
Stature	·28	Pearson and Lee	Pearson.
Span	·20	,, ,,	,,
Forearm	·20	,, ,,	,,
Duration of Life	·20	Weldon and Pearson	Yorkshire.
,, ,,	·25	,, ,,	Oxfordshire.
,, ,,	·20	,, ,,	Quakers.
General Health	·27	Ethel Elderton	Pearson.
Mean	·23		

Psychical Characters.

Intelligence	·33	Ethel Elderton	Pearson.
Truthfulness	·22	Schuster and Elderton	Heymans & Wiersma.
Temper	·18	Ethel Elderton	Pearson.
Excitability	·11	,, ,,	,,
Sympathy	·15	,, ,,	,,
Reserve	·27	,, ,,	,,
Neglect of Duty	·20	Schuster and Elderton	Heymans & Wiersma.
Tone of Voice	·26	,, ,,	,, ,,
Mean	·22		

Pathological Characters.

Insanity	·06	Goring	All Poor.
,,	·35	,,	Well-to-do Poor.
,,	·30	Pearson	Middle Classes.
Phthisis	—·01	Goring	All Poor.
,,	·16	,,	Well-to-do Poor.
,,	·24	Pearson	Middle Classes.
,,	·28	Pope	,, ,,
Of Phthisical Stock	·30	Pearson	,, ,,
Mean	·21		

Special Features.

Insanity in one mate, phthisis in the other	—·01	Goring	All Poor.
Wife of one grave, with husband of next grave	·00	Pearson and Weldon	Yorkshire.
Alcoholism	·70	Goring	All Poor.
,,	·58	,,	Well-to-do Poor.
,,	·27	Ethel Elderton	Middle Classes.

other in the psychical as well as the physical characters. Common environment can hardly give husband and wife the same eye-colour, nor the same stature, nor length of cubit—these were determined before marriage. Yet their resemblance in these matters is about the same as in their temper or temperament. Secondly, if resemblance in tuberculosis be due solely to infection, how are we to explain the resemblance in eye-colour or insanity? Are these due to infection also?

Thirdly, I recall the very remarkable point we have already considered. Dr. Goring, as we have seen, has worked out the degree of resemblance with regard to phthisis between husband and wife, among all poor and among the better class of poor. Adding my result we have the following:

Husband and Wife			All Poor	−·01
,,	,,		Prosperous Poor	+·16
,,	,,	(Pearson)	Middle Classes	+·24
,,	,,	(Dr. Williams)	Professional Classes	+·28

It would thus appear, either that like mates with like more commonly in the more intellectual classes, or that infection is more likely to occur in middle class than in poor households. I think there is not the least doubt that much of the relatively small resemblance of husband to wife in the matter of phthisis is due to a selective influence and not to infection at all. This selection is largely an intellectual one and has no existence among the very poor.

The matter is so important that we determined to take out from our family records all pairs of husbands and wives and then inquire how far phthisical stock is attracted to and mates with phthisical stock.

I collected over 220 cases of families and determined in which stocks cases of phthisis had occurred, and in which they had not; in every case the husband and wife, whether phthisical or not, were themselves omitted from consideration. Table VIII is the result.

TABLE VIII. INTERMARRIAGE OF TUBERCULOUS STOCKS.

Actual:

	Husband's Stock		
Wife's Stock	Sound.	Tuber-culous.	Totals.
Sound	588	140	728
Tuberculous	177	95	272
Totals	765	235	1,000

Correlation 0·30

Chance:

Wife's Stock	Sound.	Tuber-culous.	Totals.
Sound	557	171	728
Tuberculous	208	64	272
Totals	765	235	1,000

Transfer

+31	−31
−31	+31

In other words, 62 stocks are transferred and the correlation is 0·30, as high as in the extreme case of mating with regard to phthisis. Now it is clear that neither husband nor wife was known to be phthisical at the time of marriage, and yet in the middle and professional classes phthisical stock tends to mate with its like. We are almost forced to the conclusion that the occasions in which we find husband and wife both phthisical are not—although exceeding the range of chance—due solely to infection. It seems probable that a very large proportion of what is itself not very large is due to assortative mating. Now many persons suspect all figures, and I have often heard such a statement made as 'But if you had taken any two

characters you would have found them correlated'. To test such a statement, every wife was mated with the husband on the next grave in the churchyard, and of every husband we inquired was he insane or not and of every wife was she phthisical or not. We found that there was no relation between a husband and a wife from different graves; there was also no relation between insanity in the husband and phthisis in the wife—in both cases the correlations were zero. Thus we see that if we take things in which we should anticipate no relation, none arises.

Another character in which we should expect marked marital relationship is that of alcoholism, and, as we have seen, we undoubtedly find it!

Alcoholism, Husband and Wife.

All Poor	·70
Prosperous Poor	·58
Middle Classes	·27

Here again in the middle classes 0·27 probably represents the mating of like with like, for it is about the value we have found for other characters. But 0·70 represents the 'infection' of drink as evidenced in the criminal classes. Had the relation between husband and wife come out on this scale for phthisis we should have had no doubt about attributing two-thirds of it to infection. Having shown that husband and wife do not indicate in a marked manner the influence of infection in phthisis, we now turn to the relation of parent to child.

This problem has been dealt with by myself on Dr. Rivers' data in a paper of 1907, and more recently by Dr. Charles Goring, when discussing his observations on criminals. If infection be the principal factor in

the production of tuberculosis, we should anticipate that the relation as to phthisis between husband and wife would be at least as intimate as between father and son. By the time a son reaches the average age of onset he is largely removed from close contact with his father, but husband and wife are always together. Now in my data we had only the particulars as to the parents of phthisical children and as to their brothers and sisters. You will notice that we need a fourfold table; we need the number of phthisical and non-phthisical children of non-phthisical fathers and mothers. This datum can be supplied if we estimate the number of individuals in the community who suffer from phthisis at any time of their life. We have further to make a correction for the history of the phthisical families when completed. According to Dr. Thompson 50 per cent. of a family with tubercle are ultimately tuberculous. I have taken 33%, as actually observed in incomplete families, and the 50% observed by Dr. Thompson in completed families as the two ends of my scale. I have also considered the difference in result which arises from supposing first 10% and then 13% of the community—not to die, but to suffer at some time from tuberculosis. The general conclusion is that the resemblance between parents and offspring lies between 0·40 and 0·60. Dr. Goring was more fortunate than I was. His data actually provide the number of tuberculous persons in his population, and the only corrections he had to make were for completion of family history. This he did in three different ways—(i) by including only those individuals over 14 years, and then by including only those over 23 years, and again, (ii) by taking 33%, and (iii) 50% to be ultimately tuberculous.

None of these hypotheses made any striking difference in the correlations. They are all given below in Table IX.

Now these results are most instructive. They show first that a parent—even the father—is twice as dangerous to the offspring, if the source be infection, as the husband to the wife. They show further that, if we include only persons over 14 or 23, the wife is not more dangerous to the offspring than the husband. They have both equal influence. And yet, as the wife

TABLE IX. RESEMBLANCE OF PARENT AND OFFSPRING FOR PHTHISIS. GORING.

	Incomplete History.		*Completed History.*			
	No Correction.		33 %		50 %	
	Father.	Mother.	Father.	Mother.	Father.	Mother.
All offspring	·44	·49	·52	·56	·60	·63
All over 14 years	·50	·50	·54	·54	·62	·61
All over 23 years	·50	·52	·54	·54	·62	·62

is closer to the husband than the father to his children over 23, and the mother to her children than the father, the sole influence of this superior infection occurs when we include children under 14! The father is twice as dangerous to the child as the husband to the wife! The mother is only very slightly more dangerous than the father at very early ages! It seems to me that we have run up against a stone wall of fact which no theory of infection can batter down! If the main factor be infection why should the father and mother be equally influential with their children, and why should the father be twice as influential as the husband? I cannot get over this wall of fact. It has

for me only one possible explanation: the hereditary constitutional factor is immensely more important than the infection-factor, and when we devote all our national energies to isolation and segregation, we are wasting a very large proportion of our efforts. I have selected two tables—one from my own work and one from Dr. Goring's, deduced by quite different methods, with wholly different hypotheses and using absolutely unlike material. Both are reduced to 1,000 cases.

TABLE X. ILLUSTRATIVE TABLES.

PEARSON: Parent and Child.

		Parent Not T.	T.	Totals.
Child	Not T.	847	23	870
	T.	107	23	130
Totals		954	46	1,000

Correlation 0·55

Transfer

+17	−17
−17	+17

GORING. Father and Child.

		Father Not T.	T.	Totals.
Child	Not T.	869	46	915
	T.	62	23	85
Totals		931	69	1,000

Correlation 0·52

Transfer

+17	−17
−17	+17

In both cases the tables correspond to a transfer from the independent probability of 34 individuals, as against the 6 in the case of husband and wife. The correlations are respectively 0·52 and 0·55. I think these results should convince you, as they have already convinced me, that tuberculosis has not to be considered solely from the standpoint of infection—that the constitutional or hereditary factor is of far greater significance.

Now let us look at the general inheritance—the resemblance between parent and child—for physical characters as shown in Table XI.

These results suffice to indicate that the intensity of parental resemblance with regard to phthisis is absolutely similar to what we find for insanity or deaf-mutism, which certainly do not arise from infection, and is of the same order as we know occurs in the case of the chief physical characters in man.

TABLE XI. INHERITANCE OF CHARACTERS IN MAN.

	Character.	Correlation.	Authority.	Data.
Physical	Stature	·51	Pearson & Lee	Pearson's F. R.
	Span	·46	,, ,,	,, ,,
	Forearm	·42	,, ,,	,, ,,
	Eye Colour	·50	Pearson	Galton's F. R.
Pathological	Deaf-mutism	·54	Schuster	Dr. Fay's
	Insanity	well over ·30[1]	Pearson	Dr. Diem's
	,,	·53	Heron	Dr. Urquhart's
	,,	·47	Goring	Convict Prisons
	Phthisis	·50	Pearson	Dr. Rivers'
	,,	·50	Goring	Convict Prisons

I turn now to the next stage in my argument.

If we admit, it will be asked, the view that tuberculosis is due to the tubercle bacillus, and that this does not arise *de novo*, then surely the transmission of the bacillus—infection—must be the source of all phthisis. But who has ever denied this? In my very first paper on the subject I wrote:

'The discovery of the possibility of phthisical infection has led, I think, to under-estimation of the hereditary factor. Probably few individuals who lead a moderately active life can escape an almost daily risk

[1] Incomplete family histories.

of infection under urban conditions, but in the great bulk of cases a predisposition, a phthisical diathesis must exist, to render the risk a really great one.'

Notwithstanding that distinct statement of my position, a leading medical member of the Sociological Society believes it possible to confute the observed facts with regard to phthisis recorded in this Laboratory by the asseveration that biometricians measure everything but life and that the Director of the Biometric Laboratory has no knowledge of the tubercle bacillus! Other medical critics—possibly of more weight in the profession—seem equally at sea when they have to deal with the interpretation of the most elementary problem in statistics. Dr. Leslie Mackenzie seeks refuge in the suggestions[1] that the paternal germ-cell may carry the tubercle bacillus to the offspring and that such early infected cases may remain latent for twenty or thirty years after. 'I do not fabricate hypotheses,' wrote Newton, and the business of the trained man of science is not to suggest what may or may not be, but to accumulate facts and find out what actually is. The tubercle bacillus is not invisible; let the man of science turn to his microscope, and not scatter hypotheses broadcast in popular journals, where they pass for proven facts.

Still another eminent medical man in high official position asserted that I went beyond my data in stating that 'few individuals who lead a moderately active life can escape an almost daily risk of infection under urban conditions'. Here we come to the very kernel of the matter:—if the tubercle bacillus be ubiquitous, the difference between man and man cannot be a question of

[1] *Sociological Review*, vol. iii, p. 308. If the suggestion were demonstrated, the remedy would lie in the celibacy of the tuberculous, not in the separation of parent and offspring after birth, i. e. it would be *eugenic*.

infection. Nowadays, 8 to 10% of people die of tuberculosis; perhaps another 3 to 5% are seriously inconvenienced by it in one form or another. Is it mere chance infection that this 10–15% are selected from their fellows? Clearly the question of the prevalence of the infection, and effective infection, is a fundamental inquiry. Yet you may look through the whole of Dr. Newsholme's large and popular volume on phthisis and find no real information on this all-important point. All he does is to state, without inquiry, that *I* have none. Now I find it a very useful precept never to make a statement without a solid ground of fact, but in most publications some statements will occur for which want of space precludes full evidence. Your critics invariably rush at these points—I suppose from a general experience of how common vague statements are—and then they give themselves away. It is a simple rule, and only involves such self-control as is summed up in never speaking without facts, but sometimes storing your facts. In this case the facts were not mine; they were the common property of medical science, available to all those who realize that medical literature is not confined to a few English writers. For years the Germans have made a special study of the prevalence of tuberculosis in urban populations. There has been nothing nearly so excellent done in this country nor, indeed, elsewhere. At first, by careful post-mortem examinations, Hannau, Schlenker, Burkhardt, and Nägeli have shown that at least 50% of adults who die from other causes have suffered from latent or healed tuberculosis. Hamburger and Sluka found that 47% of all children between 11 and 14 showed healed tuberculosis, and that no less than 77% of children of the same ages showed some form of tuberculosis. Since nothing like 30% die of tuberculosis, this

means that another 15% to 20% will heal in time. Even this large number, shown by autopsy, was soon to be exceeded by von Pirquet's introduction of the tuberculin test. He himself found by cutaneous reaction 55% of children were tuberculous. Quite recently, Hamburger and Monti, following the cutaneous test by an injectional one, have found 94% of Viennese children between 11 and 14 years to be tuberculous. They further state that they have so far found no case of positive injectional reaction which has not shown tubercle on autopsy.

TABLE XII. PERCENTAGE OCCURRENCE OF TUBERCULOSIS IN CHILDREN

as determined by

Year of Life.	Autopsy.		Tuberculin Reactions.			
	Non-Lethal and Lethal.	Non-Lethal.	Cutaneous.			Cutaneous and Injectional.
	Ghon.	Ghon.	V. Pirquet.	Ganghofner.	Hamburger & Monti.	Hamburger and Monti.
2nd	40	17	2	12	9	9
3rd and 4th	60	30	13	27	23	27
5th and 6th	56	34	17	47	36	51
7th to 10th	63	35	35	57	47	71
11th to 14th	70	53	55	70	51	94

Now 94% of children between 11 and 14 do not die, even in Vienna, of tuberculosis! The great bulk of them recover and live to die of other complaints. But the existence of this enormous mass of infected children is, I think, sufficient to demonstrate the ubiquity of the infection. Why in one of these children does the disease develop to full intensity, but in another heal and the child probably become immune? I think that those who have

really thought the whole matter over carefully will recognize that the *soil* has to be considered as well as the bacillus. Does every one of those 94% of Viennese children have an actively tuberculous father or mother? Clearly and obviously not. Infection from parents cannot be the chief source of the spread of the disease among children. Whatever may be the state of affairs in the future, to-day we are compelled to regard tuberculosis as an almost ubiquitous disease through which the great bulk of the urban population passes in childhood, and having regard to the broad facts of heredity, as I have laid them before you, to hold that certain stocks are relatively more immune [1] than others, or acquire this immunity more readily as a result of attacks in childhood.

Let us now consider for a moment that a population is composed of stocks with varying degrees of immunity and that it is subject to the presence of an infectious disease. Whither does this idea lead us? Clearly each case will infect a certain number of others, and these again will spread the infection to another circle. In each circle those beyond a certain intensity of immunity recover, those below it die. There is thus a selection of the less immune at each stage of the disease, and the population by the law of heredity, since the non-immune die earlier, is left gradually more and more immune. I am taking here the case of constitutional immunity, not acquired immunity. Now the whole problem thus stated admits of mathematical treatment on the basis of the theory of

[1] Two such high authorities as Drs. Newsholme and Bashford have recently denied flatly the existence of relative immunity in animals; but careful German work seems to be indicating the existence of this relative immunity in both mice and men, and finding out in what characters it can be summed up.

probability. If we start with a given population and a given number of centres of infection, we find the number of cases either tends to increase or to decrease according to the ratio of cases to population, but after a certain period approaches a finite asymptotic value—what we may term the stable value.

The history of an infectious disease is represented by a diagram of this kind:

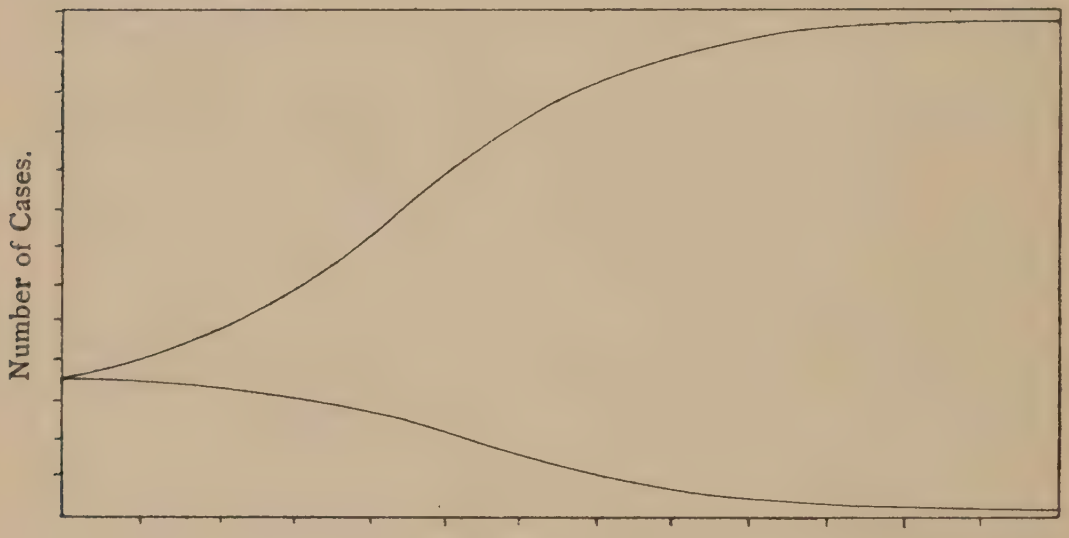


Fig. IV.

It does not tend to increase or decrease indefinitely. Which case arises depends on the proportion of centres of infection to total population at the start. The number of cases then remains stable until some marked change in environment or ratio of population to infection-centres starts the process afresh.

Now something precisely of this kind, this 'natural history' of an infectious disease, is to be expected in the case of tuberculosis, if there be a real hereditary immunity. Do we find it?

To answer this let us look at the diagrams for male and female deaths. The first diagram represents the general death-rate corrected for age distribution in the population.

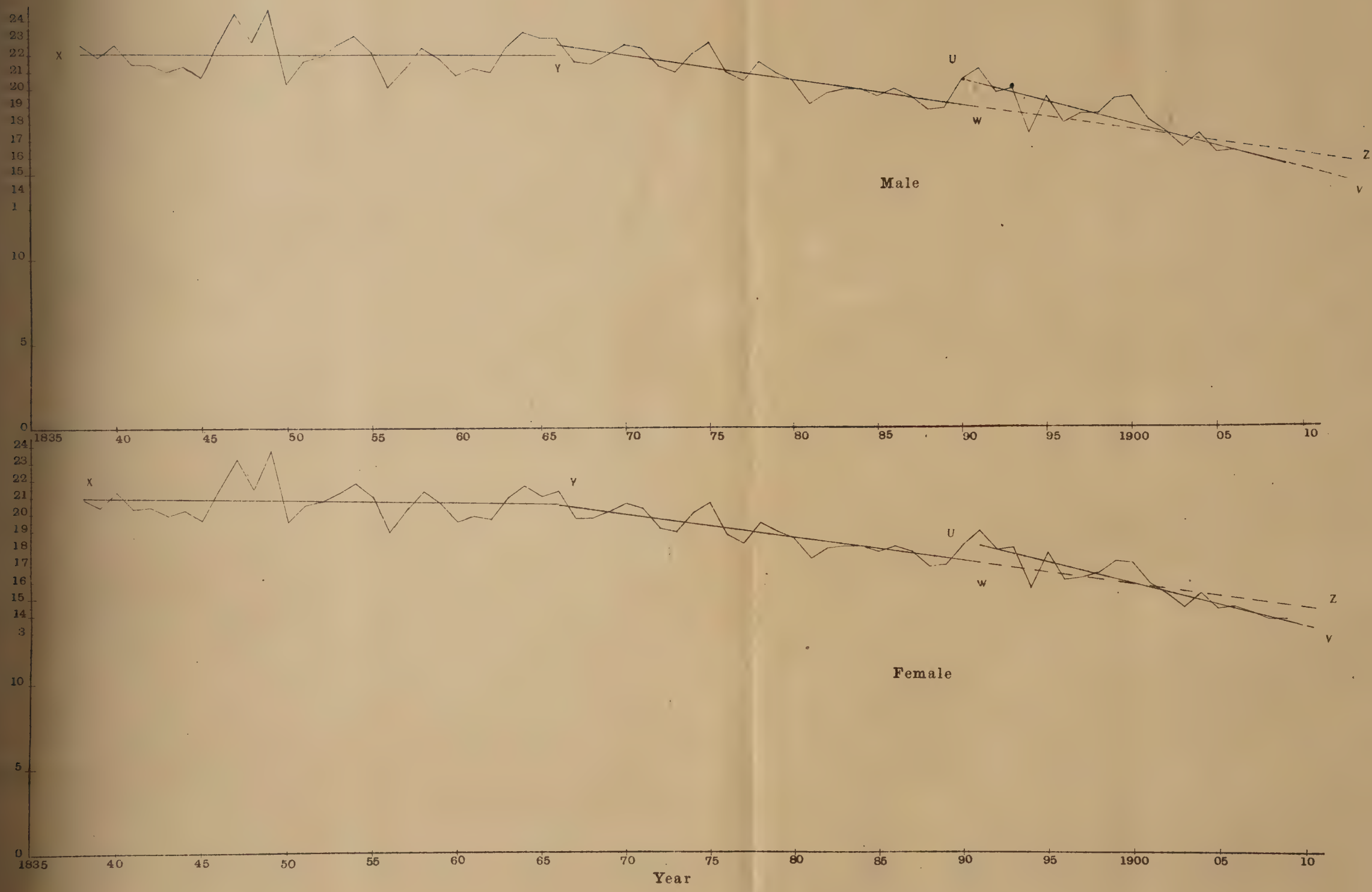


Fig. V. The Fall in the General Death Rate

I owe the data to the courtesy of the Registrar-General. On inspection you will see that the history of the general death-rate in this country consists of three chapters. Chapter I (1835–66). In this period of 30 years, there is no appreciable diminution in the death-rate of either men or women. But this is the great period of factory legislation! That legislation was brought about by the Reports of Royal Commissions, 1820–45, showing the terrible condition of the work-people. There is no evidence to indicate that the factory legislation in any way improved the national mortality. Chapter II (1866–91). This period of 25 years is essentially that of sanitary reform. It is not my purpose to determine now whether that reform produced the marked fall we now note in the death-rate. What is quite certain is that, allowing for age changes in the population, the death-rate did fall in those years in a marked way for both men and women. Chapter III (1891–1910). This last period of 19 years is the period of modern medicine, the period during which there have been two ideas always present —that of the local medical officer and of bacteria as the origin of disease. If you examine the curves, you will see at once that there at least is association, if there be not causation, between the presence of these two factors and an accelerated fall in the death-rate. Let us all rejoice in this fall, and for the present and, perhaps, ultimately with greater knowledge give due credit to sanitary legislators and increased medical knowledge. The whole diagram is satisfactory; we have increasing rate of fall in our national mortality.

Now I proceed to an examination of Fig. VI, which gives the death-rates from phthisis corrected for age. We see at once that the whole sweep of the curves is different. To begin with, there is no early

period of quiescence or stability. Before 1847 the scanty data available seem to indicate an extraordinarily rapid fall in the phthisis death-rate. During our first period from 1847 to 1866, instead of stability we notice that the death-rate from phthisis fell long before the general death-rate, and before what I have ter ed the period of sanitation. This in itself indicates a natural rather than an artificial decay of phthisis. During the sanitation period (1866–91) the fall in the phthisis death-rate has been more marked than in the general death-rate, and most noteworthy decreases took place. Now we come to the last period involving three things (1891–1910) —the discovery of the tubercle bacillus, the introduction of sanatoria, and the so-called Fight against Tuberculosis. Please regard my diagrams carefully here. To begin with, they are made out without a knowledge of the census returns for 1911. What will be the effect of this knowledge when it comes to us? Now it seems to me that of two things we may be certain—(i) that the estimated population will exceed its true value; (ii) that the population in actuality will be more adult than its estimated distribution,—it will have more individuals in the phthisical ages. Now what does this mean? I think it must indicate that the known number of phthisical deaths have to be divided by a lessened exposed-to-risk population, but that this will be slightly counteracted by the age averages. In other words, our rates for the years 1900 to 1910 are almost certainly *too small.* I think we may safely say that our curves do not exaggerate but minimize what has been taking place, namely, that during the years of tubercle bacillus, of sanatoria, and of the fight against tuberculosis, the rate of fall in the death-rate from phthisis, instead of being accelerated, has been *retarded.*

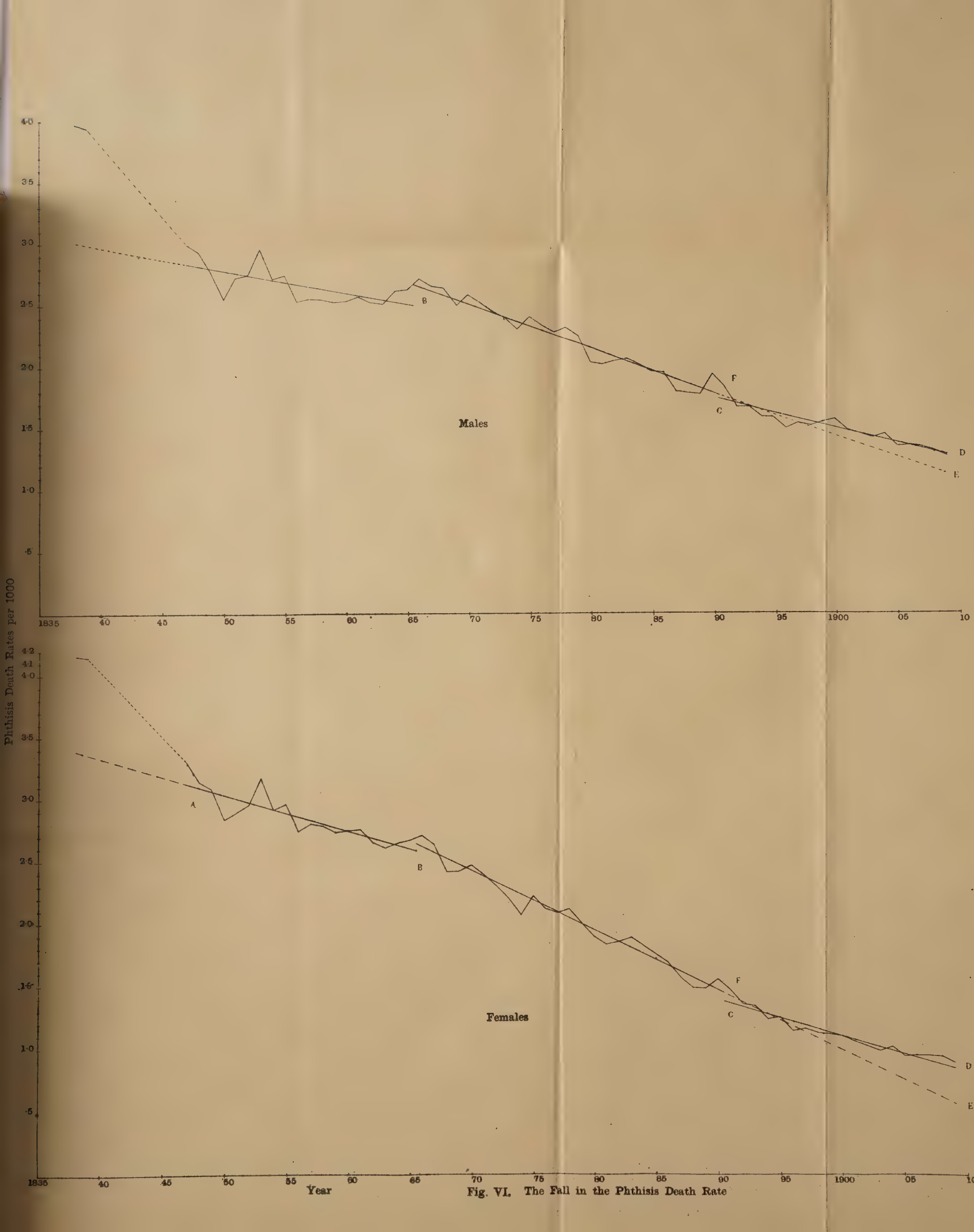


Fig. VI. The Fall in the Phthisis Death Rate

Examine the same phenomenon apart from population totals, namely, by considering whether fewer deaths now occur from phthisis relative to all deaths. See Fig. VII, p. 30. During our first period we see a very sensible drop in the relative phthisis death-rate; then with the sanitation period comes the accelerated fall, and then in our last period of greatest activity—in the last 15 years, when we are said to know all about phthisis, and when we are told that a sufficient number of millions have only to be spent in a certain manner in order to abolish it altogether —we find—what? That the phthisis death-rate relative to all deaths has begun to rise in males, and is almost constant in females, or indeed, since 1900, has also begun to rise.

Now, Ladies and Gentlemen, various explanations have been offered to me of the source of this retarded fall in the phthisis death-rate. The first I give you is certainly ingenious, and may have played some part in the matter. Formerly, the constitutional factor was recognized by laymen and medical men as an important factor, and marriages with or between phthisical stocks were everywhere discouraged. Then came the swing of the pendulum: the bacillus was the only thing to be regarded, and it did not like fresh air. The only thing needful was to keep bedroom windows open, and marital selection was of no importance. The result, according to this suggestion, has been increased panmixia and a production of stocks constitutionally liable to lethal tuberculosis in larger numbers.

A second explanation has been given me by a medical friend. His view is that the increase is due to better diagnosis of lethal tuberculosis in our last period of improved medical knowledge. The question is whether the average general practitioner, who gives the great

mass of death-certificates, would to-day, owing to his better education, attribute to tuberculosis deaths which 15 years ago he would have attributed to other things? But it is not sufficient that he should attribute *some* deaths now to tuberculosis which 15 years ago he would not have done; that *some* must be so great that (i) it swamps all the deaths that he formerly attributed to phthisis and now knows not to be phthisis; (ii) that it swamps also the natural fall in the phthisis death-rate and the great artificial fall said to be due to the fight against tuberculosis.

Now remember that we are not dealing here with the question of whether persons in an *early* stage of illness have or have not suffered from phthisis, but whether persons who have actually *died* under care will nowadays be returned in large numbers as dying of phthisis, whereas 15 years ago they would be said to have died of something else. The numbers must be very large indeed, for if you look at the rate for women under the old dispensation, the death-rate should now have fallen to about 6 per 1000, while it actually is about 9. In other words, an increase of 50% of deaths of women must be due to improved diagnosis of phthisis! I cannot accept this for a moment, and for two very good reasons: (i) because I found, on going into the statistics, that improved diagnosis, at any rate at an early stage, seemed to tell quite the other way, i. e. that a considerable number of cases, 10 to 20%, considered as *possibly* phthisis were on fuller investigation found to be something else;[1] and secondly, because I had taken the precaution of consulting one of our chief authorities

[1] That there is no great excess of accuracy even nowadays in the ante-mortem diagnosis of phthisis is evidenced by the following results taken from a paper by Richard C. Cabot, M.D.: 'A Study of Mistaken Diagnoses,'

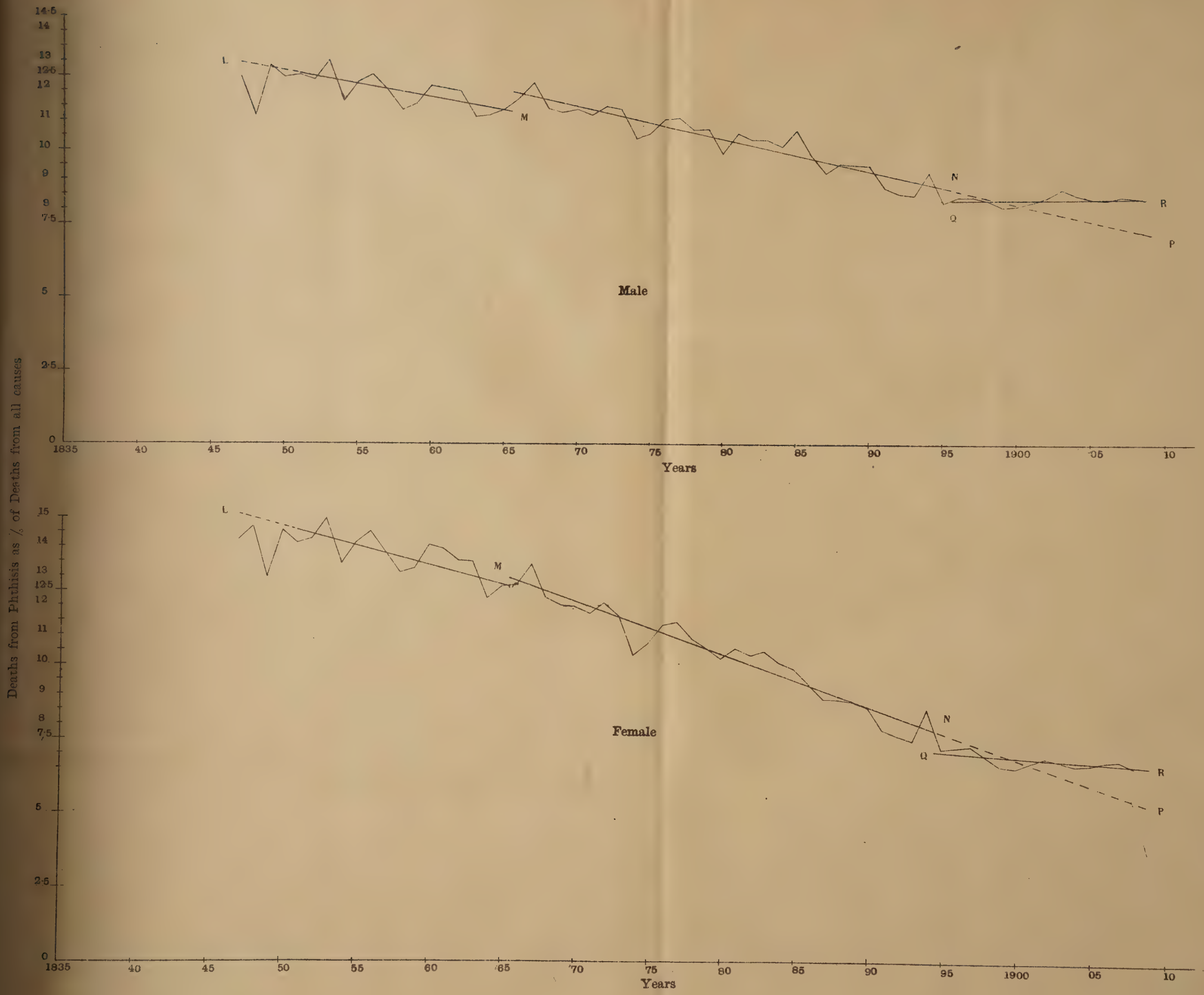


Fig. VII. The Fall in the Percentage of Phthisis Deaths and the check to the Fall since the Tubercle Bacillus has been discovered

on the subject before writing a word myself in the matter, and his view had been quite definite on this very point: 'There has been no sensible change in the conditions under which a medical man diagnoses a death as due to phthisis in the last 15 years.'

The last argument I have heard used is the curious one that the diagnosis of lethal tuberculosis itself is so faulty that the Registrar-General's death-rates from phthisis are of no value for any purposes at all! That is, I think, the last wriggle of those who dislike the broad conclusion that modern advance in our knowledge of tuberculosis has not been accompanied by increased fall in the phthisis death-rate. But if it were true, what then? Why, the whole of the so-called 'Fight against Tuberculosis' is based on erroneous premises, for that Fight has proceeded on these very statistics—namely, by the assertion that there is a fall in the phthisis death-rate and that it has been produced by the application of our increased knowledge!

Publications of the Massachusetts General Hospital. Medical and Surgical Papers, vol. iii, October, 1911, No. 3, p. 406 :—

Phthisis (Active).

Correct diagnosis in	32 cases or 59 %
Error of omission in	16 cases or 30 %
Error of commission in	6 cases or 11 %
	54 cases

Miliary Tuberculosis.

Correct diagnosis in	28 cases or 52 %
Error of omission in	20 cases or 37 %
Error of commission in	6 cases or 11 %
	54 cases

Numerous cases of obsolete or healed phthisis were left out of account wholly.

'The 30% of unrecognized cases was made up mainly of cases seen for the first time within a few hours of death, when all distinctions are blurred,' p. 407. Still in the cases discussed above we are dealing with the highest diagnostic skill—one far above the average of that of the general practitioner. It will be seen how serious is still the error of omission.

If, however, we accept the view that there is a real retardation in the fall in the phthisis death-rate, is that not precisely what we should anticipate, if we attribute the change in death-rate to the natural increase of immunity due to a selective death-rate acting on a community with varying grades of hereditary immunity?

Let us see if there is anything to support this view. If the view be correct, we should anticipate that the fall in the death-rate is largely independent of all the crusades against tuberculosis. There are various methods of carrying on the crusade against tuberculosis—the supporters of individual methods believe them alone to be efficient, and scoff at all others; the leaders of the medical profession state that they believe all to have their proper place on occasion, which may be true but does not help us very much,—and there are certain doubting Thomases, who would like careful inquiry into the efficacy of each one of them as a preliminary step to real knowledge. Well—omitting our old friend, cod-liver oil—there are (*a*) tuberculin in a variety of forms and doses; (*b*) the system of local visitation and dispensaries; and (*c*) the sanatorium 'cure'. To what extent have one or all of these reduced the mortality from tuberculosis, or increased the longevity of the tuberculous? Well, of tuberculin I propose to say nothing to-night. I have absolutely no knowledge and therefore no opinion. But our Laboratory is at work on the data for many thousand cases, and in another twelve months it will be possible to speak more definitely on the subject. Next I will take the Sanatorium influence on tuberculosis. This is a very thorny topic to treat towards the end of a long lecture, and if I make sweeping statements without ample proof, it is not absence of proof but want of time

which stands in my way. To begin with there is now a large capital involved in sanatoria, there are big staffs employed, and much of their income depends on appeal to the public. The result is that hardly a single sanatorium in this country publishes a report from which it is possible to extract information of scientific value as to their efficacy. You must either get data behind the reports — and this is often very difficult because in nine cases out of ten there exist no proper records, even unpublished—or you must seek material from foreign sources. Owing to the labours of two well-known actuaries — Mr. W. Palin Elderton and Mr. S. T. Perry—we are now in a position to give a first report on both English and American experience. The only scientific method of investigating the problem is to obtain the mortality rate in each age-group of those admitted into sanatoria, and then to compare this mortality rate with (i) that of the corresponding age-groups in the general population, and (ii) that of the corresponding age-groups of the tuberculous in pre-sanatorium days.

Now the patients may be classified by their state on admission as:

I. Incipient Cases,
II. Moderately Advanced Cases,
III. Far Advanced Cases;

or, by their condition on discharge as:

I. Apparently Cured,
II. Arrested,
III. Active.

In the case of the English data Messrs. Elderton and Perry had 7–8 years of post-sanatorium experience to judge by; in the case of the American

data, 19–20 years; and in the case of the German, 4–5 years.

The number of actual and expected deaths for each year of experience were ascertained and summed, and the general conclusions are given in the accompanying table:

TABLE XIII. DEATHS AFTER TREATMENT.
PHTHISICAL CASES.

ELDERTON.

Observer and Nature of Treatment and Case.	Ratio of Actual to Expected Deaths. Life Table (6).
In sanatorium Days—	
Bardswell.	
On Admission — Incipient	4·1
On Admission — Moderately Advanced	7·8
On Admission — Far Advanced	37·5
On Admission — All	13·1
On Discharge — Apparently Cured	2·0
On Discharge — Arrested	4·4
On Discharge — Active	27·7
On Discharge — All	12·0
Brown and Pope.	
On Discharge — Apparently Cured	3·0
On Discharge — Arrested	13·1
On Discharge — Active	39·5
On Discharge — All	15·9
Rumpf.	
Stage I	1·7 to 2·0
Stage II	4·5 to 5·0
Stage III	15·0 to 30·0
All	8·0 to 13·0
In pre-sanatorium Days—	
Pollock.[1]	
(1) All cases from onset	1·8
(2) All cases, neglecting duration over a year	5·4
Williams.[1]	
(1) All cases from onset	2·9
(2) All cases, assuming one year from onset to treatment	3·3
(3) All cases, assuming two years from onset to treatment	3·6

[1] Not exactly comparable with each other or with sanatoria data, but sufficient to show that there is no great improvement due to change of treatment.

For example, this first entry means that among incipient cases there were in the years of experience following the sanatorium treatment 4·1 times as many deaths as in the standard life-table for persons of the same age. In the case of the far advanced, 37·5 times as many. In examining this table I must warn you that the older data from Pollock and Williams are, while the best available, not at every point fully comparable with the sanatoria data. But the gist of the matter is that absolutely no other data have so far been issued to demonstrate whether or not the sanatoria are doing any good. The argument in favour of them has been chiefly opinion and guess-work.

In the face of such a table as the above I think we may state that there is no evidence at present to show that sanatoria have in any way prolonged the lives of the consumptive in a marked or substantial manner.

I can conceive nothing more unjustifiable than a statement recently made by a writer on the *Fight against Tuberculosis* that 80% of the cases admitted to a certain sanatorium were *cured*. In the first place, there are no data in the report of the sanatorium to which he refers which justify this statistical statement. Secondly, if it were true, which it apparently is not, that 80% of patients were dismissed from the sanatorium as 'apparently cured', then the real problem is—How does the mortality of this class compare with that of a similar class in pre-sanatorium days? Mr. Elderton's table shows that in our present state of knowledge we have no evidence that their mortality is any better than it used to be. Thirdly and lastly, we have seen that 50 to 70% at least of the population have undergone one

or more attacks of tubercle and recovered—in the bulk of cases without any treatment at all. Now if by increased technical skill in diagnosis you can ascertain these cases, call them incipient, and send them into a sanatorium, you can 'cure' 80% of them, but nature has done much the same thing outside sanatoria for a long time past. Perhaps rest and good food will do it more quickly—I cannot say. But the sole test of the 'curative' effect of sanatoria must be based on those cases recognized as tuberculous in the past—where we can compare the mortality in the past and the mortality of to-day.

It has further been asserted that England is unsuited to sanatorium treatment, or that the methods adopted on the Continent are more satisfactory. Now while the German sanatorium data rarely admit of satisfactory actuarial discussion they may be compared directly with the English as showing whether there is any marked difference in their results. The following tables show that there is very little to choose between the two! In one case, of the discharged patients 57% are actually dead $3\frac{1}{2}$ years after leaving; in the other case 55·6% are dead or unfit to work in four years after leaving! I have been struck, in fact, by the closely similar results obtained by sanatoria for the same class of patients, however treated, and wherever the sanatorium is situated, when the data are such that we can apply any rational method to their discussion.

TABLE XIV. WEICKER'S DATA.

CASES DISCHARGED FROM SANATORIA, GERMANY

(admitted in the earliest stages of the disease).

7·6 %	unfit to work or dead in	1	to $1\frac{1}{4}$	years.
19·0 %	,, ,, ,,	2	to $2\frac{1}{4}$	,,
33·3 %	,, ,, ,,	3	to $3\frac{1}{4}$	,,
55·6 %	,, ,, ,,	4		,,

TABLE XV. EXPERIENCE OF BENENDEN SANATORIUM, ENGLAND

(no account apparently taken of those who died under treatment).

PATIENTS DISCHARGED.

	After $\frac{3}{4}$ year.	$1\frac{1}{2}$ years.	$2\frac{1}{2}$ years.	$3\frac{1}{2}$ years.
On full work of those traced	54 %	45 %	35 %	37 %
Untraced of whole number	29 %	41 %	35 %	35 %
On full work of whole number	39 %	27 %	22 %	24 %
Actually dead of those traced	25 %	32 %	45 %	57 %

According to another school of medical thought, the main advantage of sanatorium treatment is the teaching of sanitary habits and the segregation of the highly infectious cases. If the sanatoria have produced good in this direction, certainly no marked evidence can be seen in the death-rates during the sanatorium period, for the old fall has not been maintained. Is it not indeed cruel to go to the public with a glowing account of the curative effect of sanatoria, when after all you are conscious that the segregation or leper-house idea is what is at the basis of your movement?

But has the theory that 'the sanatorium is a school of hygiene' any real basis in fact! Probably yes, where the middle and upper classes are concerned; but hardly any whatever when the working classes are dealt with. Let the *Local Government Board Report* on Glasgow, referred to on p. 7, be consulted in this matter. Most of the sanatorium patients return to homes where real hygiene is impossible. A large percentage of such

patients return to one-roomed tenements; even if there are two rooms, there is only one room with a fire, and the patient lives there by day, resting on the 'kitchen bed', and often sleeps there at night; sometimes the patient as husband or wife shares the same bed with wife or husband and one or two children. Even the very sputum bottle is only a trap, if it has to be emptied at a sink and under a flowing tap with all the food-vessels about! Whatever district visitors may accomplish with patience and persistency in matters of hygiene, a study of the Glasgow *Report* will show how very little the sanatorium has so far achieved as a school of hygiene!

So much for sanatorium treatment. As far as the available data go, they provide no evidence that this treatment is producing marked results; they supply no refutation of the position that the fall in the phthisis death-rate is due not to the reduction of infection but to the development by heredity of a racial immunity.

I now pass to my second factor of modern treatment —the dispensary system. It is asserted that where the infection-factor is taken into account locally, each case followed up, and instructions given as to conduct and treatment, there the phthisis death-rate falls in a most marked manner. In no town has this been so completely achieved as in Edinburgh. Edinburgh has become a model for other places in this matter. All honour to the men and women who teach our countrymen to be self-respecting and others-respecting. Good will always be done on such lines. Look too at the fall in the phthisis death-rate for Edinburgh (Fig. VIII)!

Here are the diagrams given by the Local Medical Officer of Health. During the last ten years of the

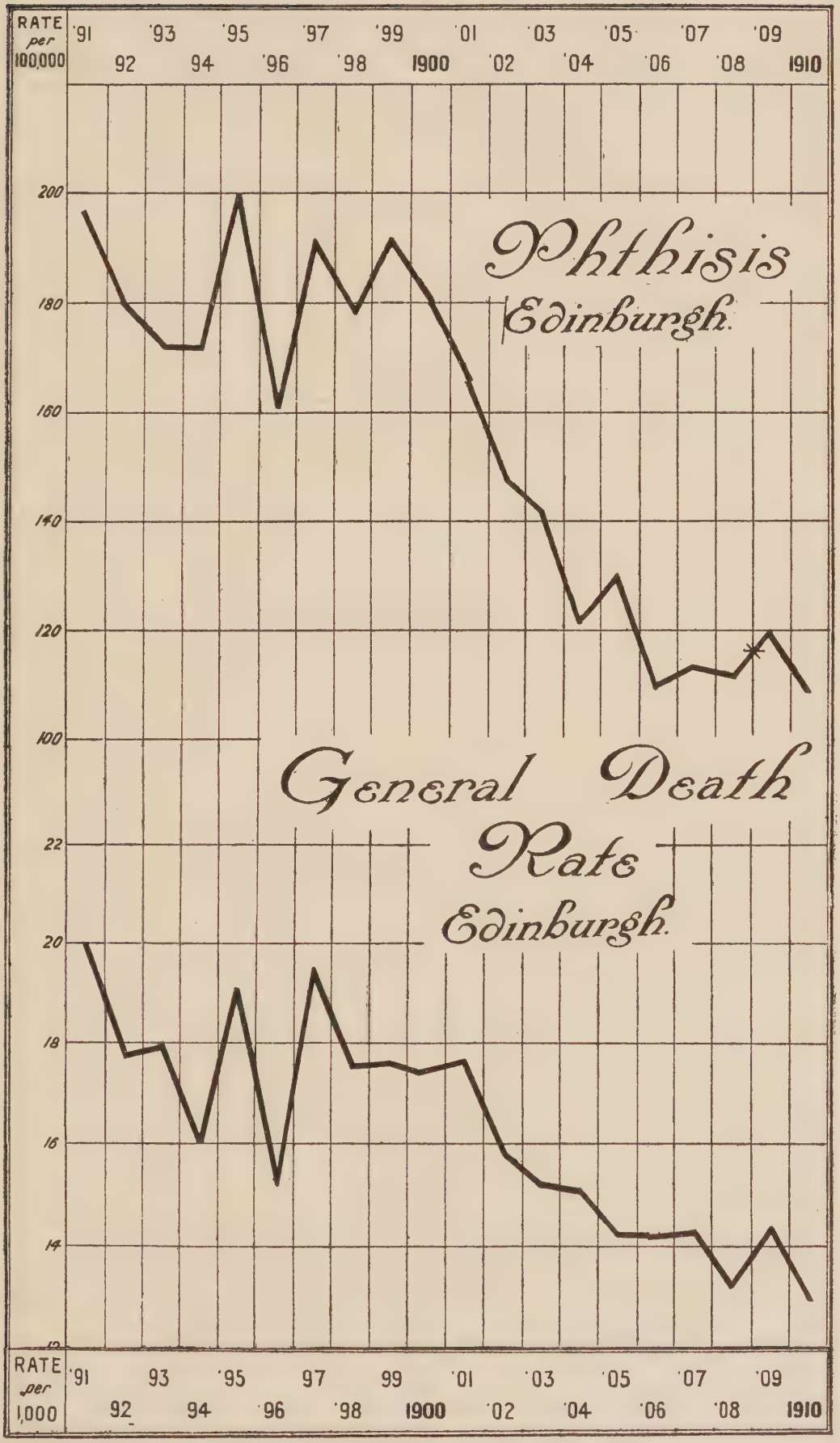
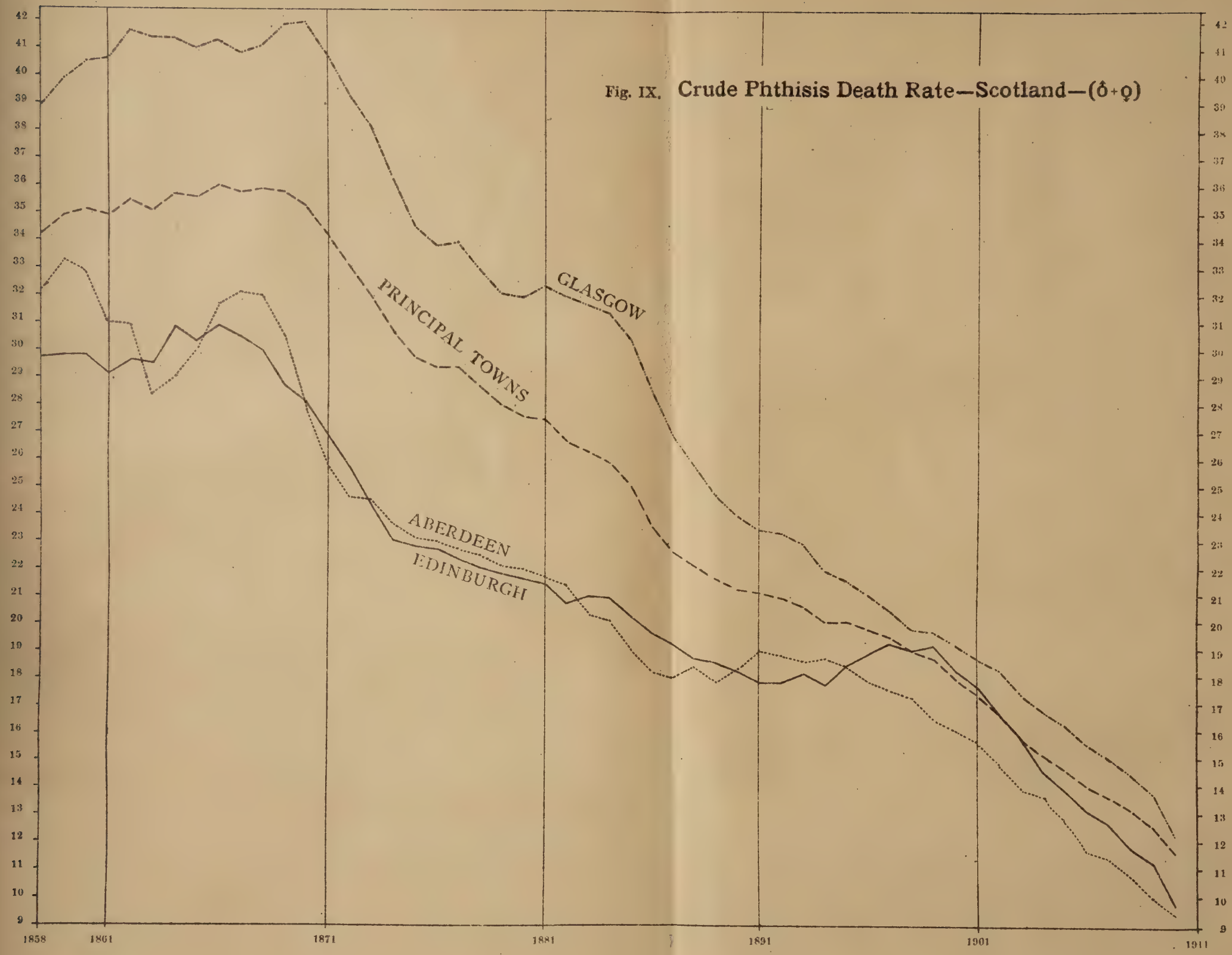


Fig. VIII. Twenty Years' Mortality.

Edinburgh dispensary system there has been a rapid fall in the death-rate from phthisis. Would it not be a grand thing to be able to point to this and say: Here is the great achievement which has resulted from this system of limiting the infection-factor? And this has been done; a recent writer asserts that this is true causation and not association, and his conclusion is at once echoed throughout the press. Well, Ladies and Gentlemen, I wish we also could accept such teaching. But it is the old tale—you take what statistics support your point and you omit consideration of the rest. The argument from statistics must always be by inter-comparison, and these great medical authorities have wholly forgotten to compare Edinburgh with other towns in which no such system of dispensaries had been established. Association, again, has been interpreted as causation.

The Scottish statistics are very bad. Scotland has done with her relatively small means such splendid scientific work, that I hope she will pardon me when I say that the data provided by her Registrar-General rank almost at the bottom of European statistics. No age-corrections are provided for the death-rates of special diseases; up to a certain date no allowances for extra-mural institution deaths have been made, and even now no allowance appears to be made for intra-mural deaths of strangers. Local officers give returns which differ from the Registrar-General's, and complete confusion, amounting often to a difference of 20%—according to the treatment of extra-mural deaths of natives and intra-mural deaths of strangers—exists between various returns. I have taken the Registrar-General's returns for Edinburgh, but these are not corrected for age and not for the deaths in the great Craiglockhart poor-house of

Fig. IX. **Crude Phthisis Death Rate—Scotland—(♂+♀)**



In order to bring out the needful features, it was necessary to smooth these curves by averaging for each five years. The crude data are very rough for single towns.

Edinburgh, which is outside the walls. Its inclusion makes the returns less favourable by about 10% for Edinburgh. But taken as they are, we see (Fig. IX) that Edinburgh occupies a position very close to the eight principal towns of Scotland, and that it would be worse than those towns if we excluded Glasgow. We see further that originally Aberdeen and Edinburgh fell in close relationship, and that now Aberdeen, without any dispensary system like the Edinburgh one, is ahead of Edinburgh. Again, although the total rate is higher in Glasgow than in Edinburgh, as we should expect in a town which collects large quantities of the general labouring class, yet the fall in the phthisis rate in Glasgow has been more continuous than in Edinburgh, if you take the whole period of our knowledge.[1] Aberdeen could with equal logic claim that an absence of the dispensary system had enabled it to produce a larger fall than Edinburgh in its phthisis death-rate. We must, I think, conclude that the fall that we find in Edinburgh is not peculiar to Edinburgh, and that if the dispensary system has produced this result there, something equally effective has produced and is producing the like effect elsewhere. The same story may be read from Fig. X, which gives the ratio of phthisical to all deaths. The fall in the phthisis death-rate in Scotland dates from long before the time of dispensaries or sanatoria. From 1868 to 1875 it was as great as it has been in the last 15 years. What logical right have we to pick out one town and one period, and say it is due to a certain treatment, whereas in other towns, where

[1] Note also (i) that the general death-rate in Edinburgh has fallen in much the same manner as the phthisis death-rate, and (ii) that for ten years the dispensary system produced no effect on the phthisis death-rate.

no such treatment exists, and in the same town before it existed, as great if not greater changes can be noted?

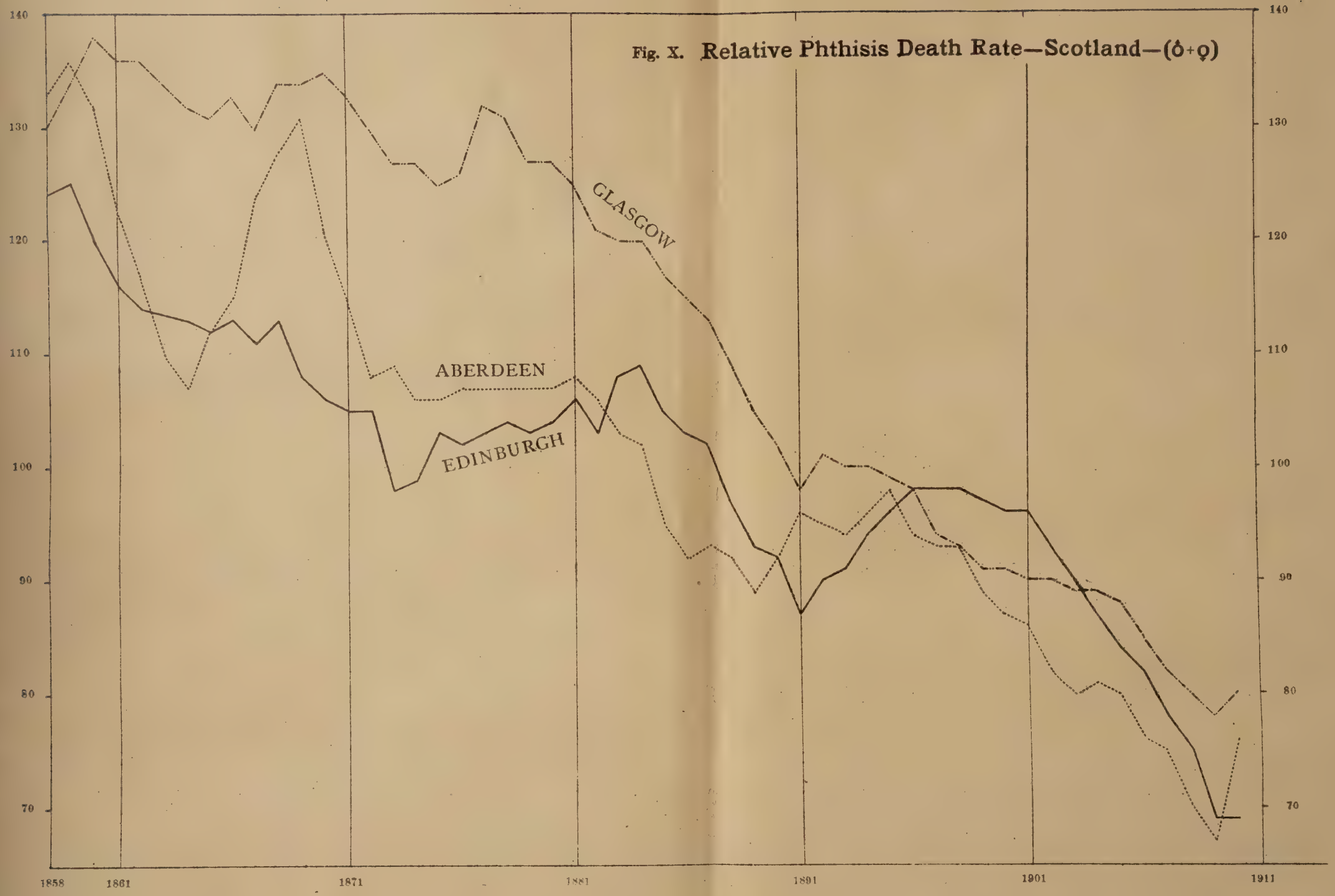
It seems to me that when we study the statistics of the fall of the phthisis death-rate, when we notice this fall taking place in urban and in rural districts, when we see that it started long before the introduction of sanatorium and dispensary work, and that it has not been accelerated by modern increase of medical knowledge, then we are compelled to regard that fall as part of the natural history of man rather than as a product of his attempts to better environment.

Does the enormous death-roll from tuberculosis during the past two centuries mean nothing at all? Without placing ourselves in the dogmatic position of asserting that every ill works to ultimate good, may we not believe that in this case human suffering has been for the benefit of the race? Once grant that in each population there are *relative grades* of *immunity* to special diseases, and that these grades are hereditary, and then we see that the natural history of any disease—which does not develop intensified virulence—will tend ultimately to stability, and in most cases to stability at a very low rate. England seems reaching that rate; Scotland is hardly yet so far advanced.

Nature, it is no wrong to assert, does more than art. Through suffering, the race has risen and will rise to more perfect physical and mental efficiency. Study Nature's methods and learn from them, and there will be response to your efforts; attribute her work to your handicraft and she goes forward with a smile; attempt to resist her progress and she will ride over you roughshod.

We of the Galton Laboratory have no axes to grind; we gain nothing, we lose nothing, by the establishment

Fig. X. **Relative Phthisis Death Rate—Scotland—(♂+♀)**



In the case of these ratios there has been no smoothing of either factors of the ratio.

of the truth that one factor rather than another is at the basis of the change in the prevalence of phthisis. Our commission is to seek for those things which tend to favour the development of a mentally and physically fit race. The easy course in life is to accept what the bulk of men are doing, and to praise its excellency. But this Laboratory would effect little if it followed, rather than attempted to lead,—if it failed to show cause for reconsideration and for knowledge before action. In this case of phthisis thousands of pounds have been spent; more thousands—indeed £1,500,000—will be immediately available, and there has been no due consideration, no knowledge, before action. If, as we anticipate—if, as I have endeavoured to show you to-night, the constitutional factor is as important as, nay, is more important than, the infection-factor, then from the eugenic standpoint a grave responsibility rests on those who assert that infection is everything, and that stocks with hereditary tuberculous diathesis have no real existence. Once upon a time the medical profession told those whom they suspected of a tuberculous tendency that it was a good thing for *themselves* to marry; then, when the existence of a constitutional weakness became a recognized faith, they told members of a tuberculous stock that it was a bad thing for the *race* that they should marry. Now the fashion has turned again, and infection is supposed to be the sole important factor, till there is danger of the tuberculous being treated as pariahs and segregated. Well, now we are told that those of stock in which tubercle has been rampant may marry as they please, provided they sleep with their bedroom windows open!

Which of these opinions is right? You may say *one*

must be. I have no hesitation in asserting that every one of them was wholly wrong *morally*—that is, socially. And for this simple reason, that they were propounded and widely taught without adequate investigation of the facts. Being right is no excuse whatever for holding an opinion which has not been based on any adequate consideration of the facts involved in it. Admit that sanatorium treatment is purely experimental, admit that dispensaries are another experiment, and that tuberculin is still another and perhaps more hazardous one, and there is nothing more to be said than the words: 'Experiment, but record your observations in such manner that the trained mind can ultimately measure their bearing on human welfare.'

But experiment on human beings is held in itself to be reprehensible. That does not mean that it is not being made day by day; it means simply that it is screened, and the experimental treatment is described as the most efficient and certain cure for human ills. Such description not only disguises its experimental character, but often hides its true nature from the actual experimenter, who forgets the necessity for adequate records to test the value of his work. That has been largely the case in the modern treatment of phthisis. I feel certain that the best medical minds of to-day would admit and regret it; they have growing doubts as to whether, after all, infection is everything and hereditary constitution of no account. They stand, however, within the ranks, and find it difficult to criticize their leaders. But now that those leaders are appealing to the public for vast funds to carry on on definite lines the fight against consumption, may we not ask for the grounds of the faith that is in them: namely, that

a campaign which pays no regard to heredity will materially accelerate the fall which has been in progress for at least 60 years in the phthisis death-rate, and which at seasons went on just as rapidly before men were familiar with the tubercle bacillus or had adopted modern methods of treatment? In the ultimate court of appeal the only evidence admissible is that of facts exhibited in adequately treated statistics, and the public must in the end tire of mere words and demand numerical data for the faith that is in the men who admit no hereditary factor in phthisis.

Dr. Lister, in a lecture delivered only a few weeks ago, spoke as follows:

> 'But the individual resistance of the patient—his relative immunity—is an unknown factor, and so statistical experts riddle the results of sanatorium treatment with criticism, while unable to tell us what better to do' (*Lancet*, March 9, 1912).

In the first place, why are statistical experts called upon to find something better to do, when Nature is solving the problem for us? It is a counsel of despair to spend millions when you have no evidence of the efficiency of the expenditure, because you have nothing better to propose. In the next place, why should we do something ineffectual because we have nothing better to do? To practise the ineffectual as if it were a proven cure checks the road to better things. Admit that there is as yet no cure for phthisis and it incites men to find one, far more actively than to praise existing 'cures'.

But Eugenists really have something better to propose. No one can study the pedigrees of pathological states, insanity, mental defect, albinism, &c., collected by our Laboratory, without being struck by the large

proportion of tuberculous members—occasionally the tuberculous man is a brilliant member of our race—but the bulk of the tuberculous belong to stocks which we want *ab initio* to discourage. Everything which tends to check the multiplication of the unfit, to emphasize the fertility of the physically and mentally healthy, will *pro tanto* aid Nature's method of reducing the phthisical death-rate. That is what the Eugenist proclaims as the 'better thing to do', and £1,500,000 spent in encouraging healthy parentage would do more than the establishment of a sanatorium in every township.

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